Elderly implies that the answers which may be produced by this large multinational, hospital-based trial will be no more clearcut than the ones we reported from our small single-centre trial. If so, we share the hopes of Professor W S Peart and his colleagues (p 1397) that a further "trial of antihypertensive treatment in people aged 65-74 may be mounted attempting to determine the balance between the benefits of pressure reduction and any adverse effects."

M E SPRACKLING

Department of Health Care of the Elderly, Sherwood Hospital, Nottingham NG5 1PD

J R A MITCHELL

Department of Medicine, University Hospital, Nottingham NG7 2UH

A H SHORT

Department of Physiology, Medical School, Nottingham NG7 2UH

G C M WATT

Glyncorrwyg Health Centre, Glyncorrwyg, W Glam SA13 3DP

SIR,—I must thank Dr Mary R Bliss (30 January, p 347) for her comments on my letter (2 January, p 50) but point out that we are writing of two quite different problems. I wrote of patients referred to me because of severe hypertension with symptoms—left ventricular failure, retinal haemorrhage, subarachnoid bleeds, and transient cerebral episodes were among them. Treatment was essential, but after years of blood pressure control I tried to discover whether the survivors, by then elderly, could be weaned from the drugs. This relates to Dr F J Flint's original question (14 November, p 1336).

Dr Bliss, on the other hand, writes of hypertensives whose treatment "may" be continued, and must have in mind the symptomless patients whom we must now aim to discover, and about whose management we have time to debate. I fully agree with her that hypotensive treatment requires most careful follow-up; but withdrawing treatment that was started with clear indications requires even more care.

C P PETCH

King's Lynn, Norfolk PE31 6HA

Primary health care in residential homes for the elderly

SIR,—While Dr M E M Herford (30 January, p 347) does not appear to enjoy an optimal working relationship with the staff of the home for which he is responsible, his point that the care staff act as "arbiters of who should see the doctor" could have far-reaching implications.

In a recent study of local authority homes for the elderly, 33·5% of the total 1154 residents were receiving hypnotic drugs.[1] Levels of hypnotic usage within the 24 homes varied from 16·6% to 54·5%. From a follow-up study six months later (which showed a similar pattern and prevalence of drug usage) attempts were made to account for these wide between-home differences in hypnotic prescribing.

We found no relationship between the number of GPs attending a given home and the level or variety of hypnotics prescribed. However, a consistent and somewhat paradoxical relationship did emerge between hypnotic usage and the resident's level of dependence. As a group, the least dependent residents (that is, those with the least degree of mental or physical impairment) showed the highest probability of receiving hypnotics, and the probability of receiving hypnotics was appreciably less in high-dependency groups. Similar findings have been reported in the USA.[2]

Various factors, acting singly or in combination, might account for this relationship. Disturbed sleep in a residential home can arise from several non-medical causes (for example, unfamiliar surroundings, sharing a room, departures from an established daily routine, etc). If such problems are, as Dr Herford suggests, selectively referred to the general practitioner, then it is possible that the prescribing of hypnotic drugs is, to some extent, mediated by the staff's perception of need. It is plausible that the needs of the least dependent, perhaps more demonstrative, residents are better communicated to the staff than are the needs of the more dependent individuals. As Dr M J Clarke and others (14 November, p 1307) have pointed out, care staff draw the attention of GPs to the needs of the resident as a relative might if they were at home.

This specific example illustrates some of the difficulties in providing primary health care for an increasingly debilitated population in what is, ostensibly, a non-medical institution. Clearly, residential homes for the elderly require special medical support. I would agree with the conclusions of Dr Clarke and others that such support should be flexible, and sensitive to the needs of both the residents and the home.

K MORGAN

University Department of Psychiatry, Royal Edinburgh Hospital, Edinburgh EH10 5HF

[1] Morgan K, Gilleard CJ. *Neuropharmacology* 1981; **20**:1355-6.
[2] Ingman SR, Lawson IR, Pierpauli PG, Blake P. *J Am Geriatr Soc* 1975;**23**:309-16.

Sinus arrest during treatment with amiodarone

SIR,—I read with interest the report by Dr Brian McGovern and others (16 January, p 160) of two cases of sinus arrest during treatment with amiodarone; but I must ask the question "Could these cases be attributed to a direct effect of amiodarone alone?" Might I direct attention to the fact that both the patients involved were also taking digoxin at the time the reported sinus arrest occurred?

The reported actions of amiodarone include potentiation of digoxin—presumably by a mechanism of displacement from protein-binding sites. One might expect this to be relevant in these instances as digoxin itself is thought to depress sinoatrial nodal activity through vagal stimulation and, in higher doses, through a direct effect on the myocardium—though, admittedly, this latter effect is thought to be due to an atrial conduction failure rather than an effect on sinoatrial nodal rhythmicity. It follows that sinus arrest and subsequent bradycardia could conceivably be attributed to the increased available digoxin in a patient taking both digoxin and amiodarone. I feel sure that Drs McGovern and others have considered this possibility, but this is by no means clear when one reads the article.

To this I might add another question: have there been any cases of sinus arrest occurring in patients taking amiodarone alone?

A K JONES

Accident and Emergency Department, Warrington District General Hospital, Warrington WA5 1QG

Retroperitoneal fibrosis associated with metoprolol

SIR,—Dr M J Mitchinson (30 January, p 347) makes two comments about the report of an association between retroperitoneal fibrosis and metoprolol. The first and major comment is that "idiopathic retroperitoneal fibrosis is usually such a slowly progressive disease that it seems unlikely to have reached an advanced stage in the 11 months of exposure to the drug." This view is incorrect. In a series of patients with retroperitoneal fibrosis associated with methylsergide therapy and treated surgically four out of 14 had taken the drug for less than one year and all but two for less than two years.[1] The period of exposure to metoprolol in this case is thus consistent with its being the culpable agent.

The second point made is that hypertension itself is a feature of retroperitoneal fibrosis. As Dr Mitchinson has so beautifully demonstrated,[2] hypertension is a late feature due to severe renal damage and occurs when there is bilateral hydronephrosis or a non-functioning kidney on intravenous pyelography. Hopefully, future reports of retroperitoneal fibrosis occurring in patients taking beta-blockers (and other drugs) will provide urea or creatinine and erythrocyte sedimentation values from near the time beta-blocking agents were started where possible. Reports of the occurrence of retroperitoneal fibrosis in patients taking beta-blocking agents for angina rather than hypertension are obviously of great value.[3]

The number of case reports of retroperitoneal fibrosis associated with beta-blocker usage is steadily growing. Details of all such cases will, I hope, be reported to the Committee on Safety of Medicines. Only in this way will it be determined whether retroperitoneal fibrosis is a rare but important side effect of beta-blocker therapy.

D W BULLIMORE

University Department of Medicine, St James's Hospital, Leeds LS9 7TF

[1] Graham JR, Suby HI, LeCompte PR, Sadowsky NL. *N Engl J Med* 1966;**274**:359-68.
[2] Mitchinson MJ. *Br J Surg* 1972;**59**:58-60.
[3] Pierce JR, Trostle DC, Warner JJ. *Ann Intern Med* 1981;**95**:244.

Dobutamine and salbutamol in cardiogenic shock

SIR,—The recent article by Dr M B Fowler and others (9 January, p 73) was a potentially important study undertaken to compare the haemodynamic effects of two β-agonists, with differing specificity for β_1- and β_2-adrenoceptors, in cardiogenic shock. However, the incorrect use of statistical methods in the analysis of the results seriously impairs scientific assessment of the haemodynamic effects of each and comparison between the two drugs.

In particular, the use of multiple *t* tests in a cross-over and repeated measures design is erroneous as it takes no account of the effect of multiplicity of such analyses on the estimates of significance.[1 2] An